

THE PATHOLOGY AND TREATMENT OF

LABYRINTHITIS

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LABYRINTHITIS - A REVIEW OF PATHOLOGY AND TREATMENT

Inflammation of the Inner Ear may be, in its origin,

- (Otogenous (Non-specific
- (Specific including (Tuberculous
- (Syphilitic
- (Meningitic
- (Haematogenous
- (Traumatic

but of these the most common by far is the non-specific type secondary to acute and chronic middle-ear suppuration (Logan Turner and Fraser 1928) and it is with this group that this review is mainly concerned.

Labyrinthitis has been known for about 200 years although it was Galen (second century) who first applied the term labyrinth to the inner ear. Mention must be made of such pioneers as Duverney (1684) who described pus in the middle and inner ear, Leschevin (1763) who noted caries of the labyrinth and petrous that went on to meningitis and Scarpa (1772) who discovered the membranous labyrinth; but it was Itard (1821) who first associated vertigo and vomiting with aural disease, and Jansen (1893) who put our anatomico-pathological knowledge on a sound basis, while the histological studies of Wittmaack (1904), Neumann (1907), Alexander (1910) and Scheibe (1912) broadened our concept of the disease and exposed some of its problems.

As regards treatment there were originally two schools of thought. The Conservative School (Wittmaack, Heine and others), the older, considered that most cases would heal if the injurious factor, especially in the middle ear, were removed, and that no drainage of the labyrinth itself would be necessary. With the methods of examination then known it was not possible to diagnose purulent labyrinthitis with certainty, but this stage passed with the publication of Barany's great work on the caloric reaction of the ear, and

the introduction of the noise-box, the rotating chair, and the caloric tests (1907).

It was then thought that this would differentiate serous from purulent labyrinthitis, producing a more active method of procedure and giving rise to the Radical School of Vienna (Holmgren, Ruttin, Barany, Hinsberg, Neumann and others) whose teaching was that an early labyrinthectomy in the purulent labyrinthitis would avert the ever threatening meningitis. However surgical enthusiasm carried them too far, and soon operation was advised in forms of labyrinthitis where less active treatment had formerly given good results, opinions which aroused opposition especially when it was realised that even serous labyrinthitis could result in complete though temporary extinction of function.

Thus came the Intermediate School of Lund who did not believe in labyrinth surgery unless meningitis was imminent, and held that early lumbar puncture would detect this soon enough for operation to be effective.

PATHOLOGY

a) Pathways of Infection.

Inflammation may gain access to the labyrinth by several routes:-

- { direct through { Round window
- { { Oval window
- { { Defect of the Bony capsule as a result of { necrosis
- { { { mechanical trauma
- { indirect through { indirect through { blood vessels
- { { lymphatics

and invasion by one route does not exclude the other, infection possibly proceeding by both simultaneously (Dean and Wolff 1934)

The Pathway according to Altmann and Waltner (1945) is determined by the predominant character of the underlying middle-ear infection:

- { exudative - infection will spread via the windows
 - { proliferative - } erosion of the capsule will tend to result
 - { cholesteatoma - }
 - { acute exacerbations - via the windows unless erosion and fistula formation has already occurred.
- (1914)

Of the windows, Ruttin and Zange (1927) seemed to find that the oval was the more often transgressed but Dean and Wolff (1934), and Sprowl (1931) favour the round window explaining that it is the weakest point in the bony capsule, and is concave and deeply placed in a bony niche near the sinus tympani and therefore more likely to harbour a pocket of pus than the oval window, although the latter is the more exposed. In practice, examination shows inflammatory reaction at both windows of the infected labyrinth and it is difficult to distinguish histologically which is the primary and which the secondary pathway, which the invasion of the labyrinth from the middle-ear and which the break-out of infected material from labyrinth back to middle ear (Altmann and Waltner 1945). Logan Turner and Fraser (1927) found evidence of this reinvasion of the middle-ear

in cases arising from capsular erosion.

Invasion by the windows tends then to occur in the acute or exudative phase and then also the prognosis is much worse, for there is little time for nature to localize the infection by plastic exudate or adhesion, as occurs in erosion of the capsule of the horizontal semicircular canal by cholesteatoma, a process extending over months or years.

Altmann and Waltner (1945) describe first of all an infiltration with leucocytes of the middle layer of the round window membrane (the membrana propria) these originating from the vascular outer layer of the middle-ear mucosa (whether the stimulus lies within middle-ear or labyrinth), although toxins or even bacteria may already have traversed the thin endosteal layer to reach the labyrinth. Decomposition of the elastic and connective tissue fibrils of this layer results, but the mucosal outer layer remains intact for a long time, or even permanently, while its deeper layers are transformed into granulation tissue with the gradual formation of an intramucosal abscess. This may open into the tympanic or labyrinthine cavity through the thin poorly vascularised endosteal layer, the granulations invading the scala tympani and thence the vestibule.

The oval window is closed by the footplate of the stapes and the annular ligament also of three layers, and there the process of invasion is very similar, the layer of granulation even forming a neomembrane to seal off the labyrinth if the footplate sloughs out, while the granulations invade the vestibule.

Invasion of the Bony Capsule may be very sudden, as at instrumental injury in the course of a mastoid operation, but is usually

very slowly produced as a result of cholesteatoma or of chronic infection and accounts for 30-55% of all cases of Labyrinthitis (Logan Turner and Fraser 1928). In the latter instances nature has time to wall off the infection, and generalised infection of the labyrinth rarely results.

Chronic infection results in necrosis of the mucoperiosteum, and absorption of the underlying bone by a rarefying osteitis, proceeding to exposure of the endosteum lining the labyrinthine lumen (Cinelli 1938). The membranous labyrinth is thus exposed to any change of external pressure and manifests this as the "Fistula Sign", and to any exacerbation of infection within the middle-ear as a result of which spontaneous vertigo will result.

In cholesteatoma (which accounts for the great majority of fistulae) the process is more a pressure atrophy or ulceration of the bone - an even slower process with still more chance of protective barrier formation.

These processes occur usually at the prominence of the horizontal semicircular canal as it lies exposed in the narrow aditus, and rarely via the posterior or superior vertical canal as a result of extradural abscess. Zange (1919) found this latter in only 3 out of 42 cases. Erosion of the promontory is still more rare.

Indirect invasion through vascular channels may occur as a result of the extensive bed of marrow of the petrous bone; and the haversian system, especially in infants, constitutes a route of direct extension of infection, some haversian canals surrounding the cochlea and some penetrating to the endosteum of the labyrinth (Dean and Wolff 1934). Examples are Mumps, and fulminating streptococcal infections where middle ear, labyrinth and meninges are infected practically simultaneously from the bloodstream (Watkyn-Thomas 1945).

However Cinelli 1938 and Dean and Wolff 1934² have demonstrated anastomosis between the mucosal and endosteal layers of the round window membrane so that vascular spread from the middle-ear to the labyrinth is not improbable.

Retrograde invasion of the labyrinth occurs in epidemic Meningitis, via the Internal Auditory Meatus chiefly and perhaps through the Cochlear Aqueduct, although Waltner (1948) has demonstrated a barrier membrane there which prevents entry of blood or pus cells from the subarachnoid space. Neurolabyrinthitis due to the pneumococcus is not infrequent (Fraser and Dickie 1920), and to the Haemophilus Influenzae rare (Nager 1914, and Smith 1935).

b) Degree of Invasion.

According to Kopetzky (1937) the initial stage of all inflammatory reactions in the ear is a protective serous or serofibrinous exudate in the perilympatic space, with irritation and hypersecretion in the adjacent endolymphatic space. This produces a hydrops labyrinthi the duration and intensity of which determines whether or not the nerve elements of the membranous labyrinth will survive. This exudate may be absorbed with little trace: it may become haemorrhagic purulent or plastic with destruction of the membranous labyrinth (Fraser 1928). Necrosis of the endosteum results with granulations arising from the capsule which undergoes a rarefying osteitis with formation of resorption spaces and perhaps of sequestra (Altmann 1945). There is, provided no complication arises or operation intervenes, gradual complete organisation of the exudate with the filling up of the hollow spaces of the labyrinth by fibrous tissue and new bone, terminating in extensive or complete obliteration of the lumen (Logan Turner and Fraser 1927), although purulent collections may remain for a long time in the connective tissue (ditto 1928).

The period required to establish complete ossification must vary in different cases.

This is not necessarily the outcome in every case however. There may be a simple dialysis of toxins through the round window membrane from the middle-ear with resultant serous exudate in the perilymphatic space, treatment however producing complete resolution: or a localised osteitis of many years standing, ^{will,} on operative removal of the chronic infection, heal with little trace, or with lowered resistance or invasion by a virulent organism, yield rapidly to suppuration.

c) Factors

The onset of Labyrinthitis as a complication of middle-ear suppuration has been attributed to several elements. Logan Turner and Fraser (1927) state that there is "no evidence, either clinical or bacteriological, to show that a particular disease or variety of organism infecting the middle-ear is more prone than another to set up a secondary labyrinth infection" but consider three possible factors - duration of middle-ear discharge (which in varying from 2 days to 45 years did not appear to be an etiological factor) cholesteatoma (present in 63% of their cases and 74% of Uffenorde's) and aural polyp (which did not seem to predispose further than by interfering with drainage). Eschweiler (1937) stresses subacute suppuration of the middle-ear in the pathogenesis of labyrinthitis and describes inflammatory processes deep in the petrosa which often perforate into the posterior or superior canal, cochlea or internal meatus.

Labyrinthitis may also be "induced" by operative interference; minor, as in removal of aural polyp. (Logan Turner and Fraser(1927) emphasise the

danger of removing polyp by uncontrolled dissection

danger of removing polyp by snare unless functional examination shows the labyrinth to be normal), or major as in the Radical Mastoid Operation, and the mortality in this form is much higher than in spontaneous labyrinthitis (36.4% compared with 2.7% Logan Turner and Fraser 1932). In such circumstances, it is probably the breaking down of nature's protective barriers of exudate and adhesions which allows infection to invade an already threatened labyrinth; or in unskilled hands some additional injury - evulsion of the stapes or fracture of the capsule. Extra care of granulations around a possible fistula need hardly be stressed.

d) Classification.

Labyrinthitis is extremely variable in the degree and severity of the symptoms it produces, and in prognosis, and attempts therefore have been made to classify cases in such a way as to direct treatment and decide prognosis. These are based on correlation of clinical picture and histological findings.

Disease may involve the bony tissues around the labyrinth capsule, especially if highly cellular, without actual erosion of the capsule (Smith 1927), and produce repeated attacks of vertigo, spontaneous or induced by head movement, with nystagmus towards the diseased side, the labyrinthine responses being normal between attacks (Cunning 1933). This has been called Perilabyrinthitis with the admission that it is not a true labyrinthine infection, yet there may be an undetected erosion of the capsule, or the phenomenon may be a toxic one with mild serous exudation in the perilymphatic space (Mackenzie 1927). Others prefer the term Paralabyrinthitis while many consider the condition primarily a Petrositis, but it is difficult to distinguish these and true erosion in the absence of a fistula symptom (Friesner 1934).

Infection of the labyrinth may be localised to a small circumscribed area, or diffusely affect the whole organ, cochlear and vestibular.

Circumscribed labyrinthitis is the result of a small localised erosion with secondary infection which has been successfully walled off by nature. It may involve either cochlear or vestibular portion, usually the latter by necrosis of the lateral canal, with attacks of vertigo. Localisation in the cochlea, producing an inner ear type of deafness in the presence of middle-ear suppuration, is rare as it is more prone to become diffuse and be complicated by Meningitis (Cunning 1933). According to Cinelli (1938) there are no reported cases of circumscribed labyrinthitis of the cochlea.

The symptoms are well described by Logan Turner and Fraser (1927).
end

The result may be

- (Arrest of the inflammation (spontaneously or as a result of treatment)
- { and healing of the capsular erosion with connective tissue then bone,
- { the function of the labyrinth being preserved.
- (Spread of the inflammation to diffusely involve the whole labyrinth.

Diffuse Labyrinthitis implies marked interference with function and hence clinically is Manifest Labyrinthitis. There is at first a hyperaemia and serous exudation into the perilymphatic space, and later a "hydrops labyrinthi" with increased albumen, proceeding to diffuse suppuration. (Cinelli 1938).

Alexander (1910) believes that the average case of purulent labyrinthitis is preceded by a serofibrinous stage.

Youngs and Lindsay (1946) believe that in Serous Labyrinthitis the membranous labyrinth remains intact and that the inflammatory changes are limited to the perilymphatic tissues with varying degrees of degeneration of neural elements up to complete loss of function, the duration and intensity determining

whether the nerve elements will survive. Others dispute the existence of serous labyrinthitis as a distinct entity, and certainly when all function is absent it is impossible to differentiate clinically between serous and suppurative unless in retrospect should function return. Logan Turner and Fraser (1927) define it as that variety of diffuse acute labyrinthitis in which a remnant of hearing can be elicited by careful testing, and further say it can be differentiated from purulent by the absence of headache and elevation of temperature (although Uffenorde denies that pyrexia would be expected in an uncomplicated labyrinthitis). Lund (1926) and Kopetzky and Almour (1929) consider the reaction of the C.S.F. as the surest guide to the type of diffuse labyrinthitis, while Luongo (1933) admits that differentiation is possible only when the decision for operation is no longer necessary. Lillie (1934) considers that the symptoms of purulent labyrinthitis subside more quickly as the end organ is destroyed - again no help in treatment.

Kopetzky and Almour (1929) find this terminology difficult to accept and insufficient as a guide to therapy - for example a circumscribed (purulent) labyrinthitis induces a diffuse serous reaction in the remainder of the labyrinth. Thus, circumscribed lesions in the capsule should also be classed as purulent labyrinthitis associated with suppuration in the middle-ear or mastoid; and the induced serous labyrinthitis is a reaction to neighbouring suppuration.

Lund (1927) prefers the term "diffuse destructive" to purulent, the latter implying the presence of pus which should demand operation, whereas a toxic labyrinthitis may also lead to diffuse destruction.

This customary division into serous and purulent "results from an attempt to classify clinically identical phenomena on the basis of pathological evidence; but the clinical criteria are inadequate to differentiate the two forms

accurately. The serous form has been considered as a collateral oedema or a toxic reaction, the infectious process being limited to the middle-ear or mastoid; but why should the barriers of the internal ear exercise such selection and exclusion of bacteria while passing bacterial products? Yet experiments have shown that a labyrinth full of pus may still give rise to caloric reaction, and that another in which there is total loss of function will recover". (Friesner and Rosenwasser 1934).

It is therefore not the form we must diagnose but the gravity of each case.

Diffuse Manifest Labyrinthitis may -

- (1. Subside with return of function, in which case it is said, in retrospect, to have been serous.
- (2. Remain localised to the labyrinth as a focus of suppuration with no return of function: as the symptoms abate the condition is designated as latent instead of manifest.
- (3. Spread from the labyrinth to produce complications.

In Diffuse Latent Labyrinthitis (it is also called Chronic Purulent Labyrinthitis) nature has successfully walled off the infection, a diffuse inflammatory process which has resulted in the total loss of function and from which there are no longer any symptoms.

There may be healing of the capsule but still endolabyrinthine spaces present, these being full of fluid and communicating directly with the subarachnoid space, or partially or completely filled with new bone: or an active infectious process may remain communicating with middle-ear or mastoid suppuration through the fistula.

Reparation here tends to bone formation in the proliferated connective tissue which fills the labyrinth cavities - the canals and basal turns of the

cochlea are often completely filled, while in the vestibule there is only an insignificant amount, the cavity being more frequently filled with connective tissue. (Altmann 1946).

Until exposure at operation there is no certain means of differentiating these, although it is of first importance to detect a purulent focus within the inner ear to avoid disaster at operation. Logan Turner and Fraser (1932) admitted that it was difficult to say when Latent Labyrinthitis merged into spontaneous cure and suggested a period of 1 year but if in doubt considered the case as Latent. In Ruttin's compensation phenomenon unequal responses from the two sides are said to indicate that organisation of the exudate is not yet complete (Cinelli 1938) but no dependence as to surgical indications can be placed on this (Friesner and Rosenwasser 1934).

The period required to establish complete ossification must vary in different cases: it has been observed six months or more after the acute manifest stage. Then it is said that Ruttin's reaction is complete - the sound labyrinth assumes a compensatory function and after nystagmus is equal after clockwise and anti-clockwise rotation. This is not obtained in every case. (Logan Turner and Fraser 1927)

On Labyrinthitis with complications, Watkyn-Thomas (1945) said that for practical purposes the problem of the infected labyrinth is a problem of Meningitis: it is remarkable how seldom labyrinth infections cause cerebellar abscess.

The circulating fluids of the internal ear having a direct connection with the spinal fluid, it would seem that Meningitis would be inevitable in labyrinthine infections. Yet of apparently similar cases, one will recover and the other prove fatal when nature's barriers fail. (Smith 1927).

That cases presenting primarily identical symptomatology differ in the widest possible degree in their tendency to become limited and localised, heal and not extend to the intracranial contents, was the conclusion drawn by Friesner and Rosenwasser (1934) upon considering the wide differences of opinion on treatment of Diffuse Manifest Labyrinthitis.

The possible pathways by which infection may spread are

- { Internal Auditory Meatus
- { Bone Erosion
- { Endolymph
- { Perilymph
- { Others

That through the channels of the modiolus into the fundus of the internal auditory meatus is the usual pathway of extension to the meninges; but not infrequently infection may reach the fundus through nerve and vascular channels from the vestibule and ampullas instead. (Altmann and Waltner 1945).

From the fundus the inflammation may spread directly and at once to the meninges; or the process may be temporarily or directly halted by adhesions and abscess formation within the arachnoidal cistern - a dangerous semilattent phase complicated by interference with the labyrinthine blood supply and consequent necrosis of soft parts.

Erosion of bone may occur, usually through the posterior vertical canal into the posterior fossa. (Blomroos 1934).

Empyema of the endolymphatic sac is described as rare (Watkyn-Thomas 1945) or not uncommon (Altmann and Waltner 1945) but the latter authors state that meningitic invasion from there is rare.

Perilymphatic spread via the cochlear aqueduct is said to occupy a minor role; and recent work (Waltner 1948) has shown that there is not a simple physiological flow between spinal fluid and scala tympani as previously supposed,

but transudation through a barrier membrane which however may break down with a sudden increase of pressure of the spinal fluid.

Perlman and Lindsay (1939) have shown that blood or pus may invade the temporal bone from the meninges via the sheath of the Facial Nerve, or along the course of blood vessels into the cochlea. The reverse may be true in extension from labyrinth to meninges.

Intracranial spread by any of these is facilitated by the poor drainage of the labyrinth and by the tendency for the mucous membrane covering of the windows to remain intact (Altmann and Waltner 1945); such complications occur only with the diffuse form of labyrinthitis since the semicircular canals have no direct connection with the meninges (Cinelli 1938).

Further, the meningeal infection is established before, or as soon as, labyrinth function is extinguished, so that the idea of a well defined intermediate stage of Acute Diffuse Labyrinthitis between circumscribed and meningitis is only partly correct, and the connecting link, extinction of function, may be missed. (Lund 1936).

Study of the cerebro-spinal fluid by Lumbar Puncture has shown that serous labyrinthitis is associated with an increase of pressure only (Kopetzky 1929); the onset of suppuration within the labyrinth is marked by an increase in cells and protein: but this increase of cells is not an absolute indication for opening as long as the clinical picture remains benign - this incipient meningitis frequently subsides spontaneously and intervention may even be harmful although a relieving operation without opening the labyrinth is often sufficient (Eschweiller 1937).

Lund (1927) however takes an extremely different view. "If one does not start with the fact that normal cerebro-spinal fluid is free of cells and that consequently from 3 to 6 cells per cubic mm. is without doubt of

pathological significance, then studies of the cytology of the spinal fluid are without value as an indication for Labyrinthectomy": while Sprowl (1931) records that "Many authors hold that 8 or 10 white cells to the field is indicative of meningeal irritation: Ruttin believes this should be raised to 40 or 50" and Friesner and Rosenwasser (1934) conclude "We have no evidence that would lead us to believe that repeated lumbar puncture and cytological study of the spinal fluid alone can give criteria that might be used as a definite index of the degree or rapidity of extension to the meninges, and so furnish the operative indication for labyrinthectomy" and that clinical phenomena are more valuable than laboratory data as indications for operation.

Lund (1936) claims that invasion of the meninges can be early diagnosed by a slight increase of temperature plus headache (and later neck rigidity and vomiting) while recording that to await full clinical signs of meningitis invites a mortality of 75%. To be always at the bedside with a puncture needle is impossible. The connecting link between circumscribed labyrinthitis and meningitis is in extinction of Labyrinthine function. Lumbar Puncture at the moment of extinction will, according to his views, exclude cases which will localise to latent labyrinthitis and continue to be uncomplicated unless these early meningeal signs are noted. If this occurs a second spinal examination is necessary, operation being indicated when the cell count rises over 6 per cubic mm., yet he himself records cases submitted to labyrinthectomy in which hearing and/or caloric responses were still present, suggesting that he laid more stress on laboratory data than clinical observation.

The type of infection is however another factor.. Traumatic or induced labyrinthitis is manifest from the start and usually suppurative. Lund believed that while labyrinthectomy was indicated as soon as function was extinct the meninges were almost always infected so that nothing was to be

gained by postponing surgery, and that even an increase in C.S.F. cells before total loss of function might indicate operation. Logan Turner and Fraser (1932) agreed in not awaiting total loss of function. Dean and Wolff (1934) however consider acute induced labyrinthitis serious enough without the menace of an additional operation unless threatened meningitis is manifest. Kopetzky and Almour (1929) advocate control by spinal fluid examination here also.

Examination of the spinal fluid is at no time so important as in Acute Destructive Labyrinthitis during Acute Otitis Media where any cell-increase demands early operation (Lund 1936). On the other hand in cholesteatomatous suppuration there are extraordinarily significant differences - the infection is milder, nature has time to build her barriers, the prognosis is better, and meningitis does not occur until some time after loss of function, and then the cell increase is slower. This only emphasises the missing prognostic importance of the very number of cells, for while an increase from 20 to 50 cells in an acute otitis media would have a fatal outcome, an increase of several thousand in a cholesteatoma would still survive.

Kopetzky (1929) contends that when there is threatened meningeal invasion Labyrinthectomy alone is not sufficient and drainage of the lesser cisterns is necessary.

That there may be a danger in the spinal examination itself has been considered. Page (1931) believed that the frequently repeated punctures advocated by Lund might break down protective barriers against the spread of infection to the subarachnoid space, or allow infection to be drawn into the spinal canal from the infected labyrinth (Smith 1927): yet no single instance has been found.

Spinal puncture will also show that there are probably two phases in the meningeal invasion - an early abacterial protective cellular reaction with no entry of bacteria, perhaps indicative of minimal infection or of high resistance of the fluid (Bloemroos 1934), and later, entry of organisms of much worse prognostic significance (Siirala 1946)

Enough has been said on attempted differentiation of serous and suppurative labyrinthitis for the reader to realise that accurate assessment with the current hearing, caloric, and rotation tests is extremely difficult, and perhaps not without danger. Donald Watson and Norton Canfield (1945) said that they never examined vestibular function if inflammation seemed probable, and Stirk Adams (1945) records a case of meningitis induced by caloric test. Van Egmond and Jongkees (1948) however do not share this extremely conservative point of view, but on the other hand avoid Barany's rotation tests on account of possible damage from the extremely great accelerations - they consider that even a healthy organ~~is~~ liable to injury.

More important is the differentiation of other similar conditions.

Petrositis, as a consequence of the perilabyrinthine inflammation, produces vertigo, but the absence of fistula symptom and the associated pain of trigeminal distribution, with perhaps later paresis of abducent and Facial Nerves and X-Ray evidence of apical suppuration should suffice to prevent confusion.

Very similar is the toxic labyrinthitis occurring years after a Radical operation, the result of seepage of toxins from the middle-ear (Mackenzie 1927): or of fibrosis of the membranous canal (Mackenzie 1951).

In Cerebellar Abscess the vertigo is not usually of sudden onset but progressive, and hearing is usually present with Weber's test referred to

the affected ear and normal labyrinthine responses: the patient does not usually lie on the healthy side and look towards the diseased side but is often ataxic and disorientated (Dean and Wolff 1934).

A localised meningitis in the pontine angle gives a picture of more variable nystagmus and vertigo than in labyrinthitis, and again hearing and vestibular function are present. It may proceed to typical meningitis.

Other conditions producing vertigo are not associated with middle-ear suppuration.

The treatment of Labyrinthitis especially of the diffuse manifest form or of that complicating Acute Otitis has been the subject of a great diversity of opinion.

Management was on the whole conservative until following the researches of Barany there sprang up the Radical School of Vienna advocating early labyrinthectomy in the majority of cases of manifest labyrinthitis. Although this was a great advance from the days when to await clinical signs of meningitis meant a mortality of 75%, it proved rather too radical an outlook to receive acceptance by most surgeons and the more frequent use of spinal fluid examination as practised by Lund led to the more moderate school. His figures, the mortality being reduced to 31%, were an immense improvement (Lund 1936)

It was early realised however that management of labyrinthine infection complicating Acute Otitis was a problem on its own.

Logan Turner and Fraser (1927) found an incidence of 0.7% in 2660 cases. Seydell (1930) reviews the opinions recorded up to his day. Most cases were considered to be serous in nature and toxic in origin, and Ruttin (1913) considered that the earlier the onset the more likely it was to be serous, with a better prognosis. Hinsberg (1926) operated on only the exceptional cases of diffuse suppurative labyrinthitis while Zange (1928) believed that even

complete absence of reaction was not an absolute indication for surgery unless in influenza.

Holmgren (1924) and Barany (1924) held the opposite view, the former performing labyrinthine drainage the moment function was lost while Barany waited till the 8th day and operated then if function had not returned. Seydell himself favoured the conservative method, and relied on immobilisation of the body as well as the head with frequently repeated lumbar punctures to decide the moment of meningeal invasion, the mastoidectomy also being delayed as he believed such trauma only favoured a spread of infection.

Logan Turner and Fraser (1932) concurred but preferred close observation for early meningeal signs during 4 weeks of rest.

Luongo (1933) records two cases and quotes the results of Hinsberg (1930) - of 11 cases there were 8 deaths with expectant treatment. Surely operative treatment could be no more dangerous? Neumann had only 1 death in 5 cases operated upon in the acute stage. Yet surgery destroys an organ of hearing which may recover, and risks opening a normal labyrinth into an infected field; on the other hand, to delay until meningeal irritation is evident is too risky a procedure, and much more so than in labyrinthitis secondary to chronic otitis.

(1936)

Lund/insisted that spinal fluid examination was at no time so important as here, and than an increase of cells was the indication for drainage.

Spira (1937) records 8 cases with only 2 deaths, these occurring in epidemic influenza. All were treated conservatively at first, with labyrinth surgery only after Lumbar Puncture showed meningeal involvement. He concludes that the severity of infection, the general condition of the patient and the result of Lumbar Puncture should determine whether surgical intervention is indicated.

The introduction of sulphonamide treatment was a great advance. Sirala (1946) quotes Hamburger as saying that in labyrinthitis (and sinus thrombosis) operations formerly indispensable were now unnecessary, and records 3 cases of labyrinthine meningitis (abacterial protective meningitis) in which Mastoid surgery plus sulphonamides produced recovery but with loss of labyrinthine function, thus avoiding the major operation of Labyrinthotomy. He concludes that sulphonamides thus seem to be able to control the meningitis and allow us to "hasten more slowly" and observe spinal fluid changes where before an hour's delay was disastrous, but that the focus of suppuration requires surgical drainage, lest later, unopened, it causes cerebral complications. Yet if organisms are found in the C.S.F. he still recommends Radical surgery.

These findings are confirmed by Hlavacek (1947) who records 7 cases all with nystagmus to the healthy side, of which 6 were treated by antrotomy and sulphonamide; there was one cardiac death during convalescence, and 2 cases showed recovery of hearing in spite of, in one of these, absence of hearing and caloric response and a spinal cell count rising to 55 per c.mm. during treatment, while 3 suffered permanent deafness: the seventh case was rather later with a cell count reaching 1307 per cu. mm. and was submitted to Labyrinthectomy.

All these were controlled by frequent Lumbar Puncture, even 5 times per day, after early relieving operation, surgery on the inner ear being deferred until it was seen whether the increase in cells would be controlled by chemotherapy. In all the cases, Labyrinthotomy would have been justified on previous standards.

(194 5)

Altmann and Waltner/say that localisation of the infection by chemotherapy

should result in a greater number of cases of healed labyrinthitis.

Penicillin treatment has made even greater advances and in the words of Rosenwasser (1949) "Some of our previously held concepts ... may require re-evaluation in the light of the early use of chemotherapy and penicillin in adequate dosage." He records one case of Acute Otitis with vertigo and absence of hearing treated by Penicillin and Sulphadiazine with complete functional recovery - a case probably of serous labyrinthitis, and comments "Whether the early employment of massive doses of Sulphadiazine and Penicillin can overwhelm and sterilize an early suppurative invasion of the labyrinth and result in a cure with return of function, is conjectural and intriguing."

His second case is of labyrinthitis with no hearing, nystagmus to the healthy side, neck rigidity and 6,000 cells plus Pneumococcus type XII in the spinal fluid, treated by Penicillin and Sulphadiazine, but after 24 hours caloric tests showed a functioning labyrinth so that meningeal invasion must have been through other channels than the labyrinth. The patient recovered but cochlear and caloric responses did not.

Commenting on Suralas cases he doubts whether any of them had adequate or early enough Sulphonamide treatment, and concludes that one's aim must be to preserve function with early antibiotics.

More recently Huber (1950) has reported two cases following acute otitis, with functional recovery after massive doses of Penicillin and Sulphonamide. The first, associated with coryza, had wide paracentesis on admission, and the same evening showed meningeal signs which resolved under therapy without lumbar puncture. The condition seemed to settle but Facial Paralysis eleven days later required Cortical Mastoid Operation. The second following perforating

injury of the drum by a straw, had paracentesis only. Both these patients had Penicillin $\frac{1}{2}$ million units on admission, then 1.8 and 1.6 million units per day respectively to a total of 19 million units; plus I.V. solumedine 2 gm per day to 12 gm and 18 gm respectively. Treatment was commenced on the third day of the otitis in each case. Surely this is the answer to Rosenwasser's query re labyrinthitis, yet the mastoid pathology required surgical intervention.

Logan Turner and Fraser (1927) found labyrinthitis occurring as a complication of chronic otitis and mastoiditis in 1.6% of 8013 cases, which including those secondary to acute otitis gave a figure of 1.4% of all 10,673 cases of otitis - compared with the 3% quoted by Uffenorde in a series of 3135 cases.

In the management of labyrinthitis secondary to chronic otitis there are again wide divergencies of opinion, particularly in the manifest type in the efforts made to confine infection to the labyrinth or if possible check its intracranial extension (Smith 1927), and recent advances in the chemotherapy of purulent infections have radically changed our attitude - instead of preventing or treating meningitis our interest has shifted to prevention or treatment of labyrinthitis and the preservation of hearing (which is all too frequently lost), or at least to increasing the number of cases of healed labyrinthitis (Altmann 1945).

The old Viennese indication of Ruttin that every case of Labyrinthitis without function demanded Labyrinthectomy to prevent intracranial spread, no longer holds; nor does the criterion of Lund that an increase of over 6 cells in the spinal fluid required drainage of the labyrinth whether or not it was functioning - there are other possible pathways to the meninges. Others recommended opening the labyrinth only in the case of definite meningitis,

while still more conservative otologists did not believe in any operation on the labyrinth at all.

Kopetzky and Almour (1929) maintained that where no threat to the meninges existed no surgery was indicated on the labyrinth per se and removal of the causative factor (chronic mastoiditis) would allow nature to heal the labyrinthine lesion ; but that where meningeal reaction pointed to threatened invasion labyrinth surgery alone was not sufficient and drainage of the lesser cisterns was also required. With Lund (1927) they believed in spinal fluid examination as a means of control.

Sprowl (1931) took a more radical view and while admitting that some cases of serous infection might recover spontaneously, advocated immediate Labyrinthectomy for fear that delay in order to differentiate the serous type might only encourage meningitis: yet admitted that if operation was delayed repeated lumbar puncture would detect extension to the meninges.

Logan Turner and Fraser (1932) believed in Hinsberg Labyrinthotomy when function became extinct and recorded a recovery rate of 97.3% in 144 cases of uncomplicated spontaneous labyrinthine disease.

On the other hand Cunning (1933) makes no mention of serous Labyrinthitis with possible recovery, but performs Radical Mastoid operation only in the later quiescent phase, at the expense of 2 instances of sequestration out of 6 cases.

The tendency thus seemed to be conservative unless there was threatened meningeal extension so that acute Labyrinthitis could be classified as operative or non-operative (Duel 1934). The former could be further sub-divided according to the result of Lumbar Puncture, Labyrinthotomy

being considered adequate if cells only were present, while translabyrinthine drainage was required for organisms and cells.

Friesner and Rosenwasser (1934) directed that each case must be considered individually more as regards its gravity than in an attempt to differentiate the serous (and possibly recoverable) from the purulent, and recognised that certain types (Traumatic, Extensive Petrous Necrosis) were more serious than others.

Further cases of J.S. Fraser were reported by Halliday (1935) and in these 8 cases of diffuse manifest labyrinthitis were treated by Radical operation and secondary Hinsberg operation 1-6 days later - with 100% recovery.

Donald Watson however declared in 1936 that he never did the Labyrinth operation then and had had no deaths for 12 years: while J.S. Fraser commented that "In most cases of labyrinthitis the hollow spaces of the inner ear were full of pus or at any rate of infective material. If you operate you use a hammer and gouge on the mastoid and the chances are that you spread infection to the posterior cranial fossa with the result that the patient develops septic meningitis", and declared that the safest thing was to do a Hinsberg operation.

In 1936 Robert Lund published further figures to prove that reliance on absence of cells at lumbar puncture averted an unnecessary operation on one patient in four - and still further reduced the mortality.

Labyrinthectomy proved a safe operation in the hands of Frenckner who had no deaths in 18 cases, his indications being total loss of function or impending meningitis. In the presence of meningitis however there was a 36% mortality.

Better observation and treatment of chronic suppurative otitis had, according to Mysel (1942), reduced the number of cases of labyrinthitis, but a more conservative attitude reduced the mortality there from 72% to 33%. He advised avoidance of surgery during active labyrinthitis unless there was threatened intracranial extension, and if so early operation (Labyrinthectomy and not just Radical Mastoid Operation) was essential: but if the condition settled, several weeks should elapse before the Radical operation.

Lillie (1944) reviewed 23 patients who had been subjected to labyrinth operations and concluded that labyrinthotomy should be reserved for diffuse suppurative labyrinthitis without extension while imminent or actual extension demanded labyrinthectomy.

"For practical purposes the problem of the infected labyrinth is a problem of meningitis The treatment of labyrinthine suppuration is entirely conditioned by your view as how best to avoid such intracranial invasion The bony labyrinth is the only bony cavity in the body where sulphonamides do not act at a great disadvantage. We know that they freely enter the C.S.F. and we know that the perilymph is in continuity with that fluid, therefore we can reasonably hope to destroy the organisms in the labyrinth by chemotherapy." So said Watkyn-Thomas in 1945. He advocated Lumbar Puncture, and if there was no sign of invasion, heavy dosage with sulphonamides, When the acute inflammation had settled Radical Mastoid operation and drainage of the labyrinth were required.

Youngs and Lindsay (1946) reported one case of chronic otitis with recurrent meningitis and stressed that labyrinthitis might be due to a petrous focus of suppuration with inadequate drainage, and added that with

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sulphonamide and penicillin to cure meningitis, labyrinth surgery was rarely indicated.

Stirala (1946) reports only one case of chronic otitis, with acute exacerbation, which responded eventually to cortical operation and sulphapyridine with later Radical mastoid operation.

That sulphonamides help to make management more conservative is deduced by Van Egmond and Jongkees (1948), who record 56 uncomplicated cases without a death, and believe that a labyrinth operation is never, or hardly ever, indicated. Cases were treated by full dosage of sulphonamide, and if there was any meningeal irritation, by the Radical operation. They also found that sulpha-drugs had not improved the prognosis of labyrinthitis with regard to function.

Huber (1950) records one case under massive Penicillin and sulphonamide therapy. The patient received 0.8 then 1.2 million units daily plus I.V. solumedine 2 gm per day for one week and had Radical mastoid operation on the 16th day when manifest signs had subsided but otorrhoea continued i.e. the acute labyrinthitis responded to therapy but the chronic otitis did not. Hearing returned.

It is on the complicating meningitis that chemotherapy has had greatest influence.

Logan Turner and Fraser (1932) reported on 50 cases of Labyrinthitis with Intracranial complication: their mortality was 74%. Bloomroos (1934) at Helsingfors reported 16 cases with 68% mortality, and Halliday (1935) 4 cases with 75%, Frenckner (1939) 39 cases with 64% while Mysel (1942) employing early labyrinthectomy and Sulphadiazine cover reduced his own mortality from 72.2 to 33.3%. Hlavacek (1947) attempted 5 cases on Radical only plus Sulphonamides but found there was a persistent rise in cells

in
necessitating Labyrinthectomy - this is/contrast to his success with Acute Otitis.
Van Egmond and Jongkees (1948) however had more success using Radical
Mastoid Operation and sulpha-drugs, and lost 6 of 18 cases - or 3 of 15 who had
adequate dosage (20%).

Induced or Traumatic Labyrinthitis is also a problem which has been
partly considered already.

Some otologists believed that as the meninges were nearly always
infected it was better to operate early, as soon as manifest signs of
Labyrinthitis appeared, and before total loss of function. Thus Logan Turner
and J. S. Fraser (1932) advocated immediate Hinsberg operation, and even
then had a mortality of 36.4% of 22 cases (compare their mortality
of 2.7% of 144 cases of Spontaneous Labyrinthitis).

Lund (1936) with his confidence in the spinal fluid as an index
believed that a cell increase before total loss of function might indicate
operation but that loss of function was an absolute indication for
Labyrinth surgery even with no cell increase in the C.S.F.

Others held that as in other forms, loss of function was the signal,
and Atkins (1927) records a successful case treated by Hinsberg Labyrinthotomy
on the 15th day when function became extinct and spinal cell count was
58,600 per c.mm., while Smith (1927), had a similar case with fatal meningitis.

On the other hand Kopetzky and Almour (1929) report one in which there
was no cellular reaction in the meninges and which went on to spontaneous
cure but with no return of labyrinthine function. The authors attributed
this to serous labyrinthitis with degeneration of the end organ by pressure
necrosis.

These however are isolated cases and even in 1942 Mysel still advocated immediate labyrinthectomy.

Three of Hlavaceks' (1947) cases could be classed thus and at least were temporarily controlled by Sulphonamides although Labyrinth drainage was ultimately required.

If one of Huber's (1950) cases is included under this heading, then massive doses of Penicillin would seem to control labyrinth and meningeal infection with return of function - provided of course that the infecting organism is sensitive.

Latent Labyrinthitis - when function has been lost as a result of inflammation and symptoms are no longer manifest - is well described by Friesner and Rosenwasser (1934). It may be classified as

- (Repaired where the capsule has healed leaving fluid spaces within, which communicate with spinal fluid.
- (Operation here would be as dangerous as on a healthy labyrinth in an infected field.
- (Ossified where operation would be innocuous.
- (Fistulous where the labyrinth is likened to a diseased cell which should be exenterated.
- (Complicated, and that may be either
 - (Spontaneous
 - (of and in either early drainage is required.
 - (Induced

Ruttin (1914) was of the opinion that Latent Labyrinthitis caused complications especially in the posterior fossa but according to Lund's (1936) figures only 5 of 47 cases had such complications yet 8 cases of cerebral abscess resulted during acute labyrinthitis.

Most otologists however believed that it was a quiescent and relatively safe condition and required no treatment per se., but that if any other ear

operation were required, Labyrinthectomy should be performed simultaneously (Logan Turner and Fraser 1932). This would hardly be a safe procedure in the repaired labyrinth and later opinion was rather that the labyrinth should be undisturbed unless a fistula into it was found at Mastoid operation (Lillie 1944).

Complications however require treatment, whether these are Facial Paralysis (evidence of osteitis of the capsule which itself requires drainage - Lund 1936, and Hilding 1937), threatened or manifest meningitis (Frénckner 1939), or sequestrum formation (Hilding 1937), and in these circumstances Labyrinthectomy is justified. Seward (1941) reports one case which responded to Hinsberg and sulphonamides. Activation by the trauma incident to Radical operation may give rise to rapid extension. (Friesner and Rosenwasser 1939)

There remains circumscribed labyrinthitis in which the hearing may be good or poor. In the former it has always been customary to allow a period of rest until the inflammation becomes quiescent, and then to remove the causative factor, usually chronic mastoiditis by the Radical Mastoid Operation, when nature will heal the lesion of the labyrinth capsule. Ruttin (1914) however held some exceptions to this in the case of meningitis, cerebellar abscess or intractable vertigo: these have since been discounted. Lund (1936) however found that 10% suffered acute labyrinth extinction and 5% intracranial complications. Tamari (1945) records 10 cases operated with Sulphathiazole post-operatively, only one being complicated by diffuse labyrinthitis which recovered.

In the presence of poor hearing however it was found safer to perform Labyrinthectomy than await possible meningitis (Halliday 1935). Lund (1936) here again advocated control by reaction of the spinal fluid and advised Labyrinth drainage with a rising cell count or an initial count of over 15-20 cells. Frenchner (1939) however waited until meningeal irritation or severe vertigo before reverting to surgery. Hlavacek (1947) records one case in which the Radical Mastoid operation induced Acute Labyrinthitis with meningeal irritation which did not respond to sulpha-drugs and in which he had recourse to labyrinth surgery.

R E S U L T S .

The following is a Report of all the cases of Labyrinthitis occurring in Wards 39 and 40, Royal Infirmary, Edinburgh, between January 1932 and December 1950 - a period of 19 Years.

There are 124 Cases made up as follows:-

	<u>No. of Cases.</u>	<u>No. of Deaths.</u>	<u>%</u>
CIRCUMSCRIBED.....	43	0	0
MANIFEST { Spontaneous } ..	52	9	17.3
{ Induced } ..	11	2	18.2
LATENT.....	18	0	0
	<hr/> 124	<hr/> 11	<hr/> 8.87 <hr/>

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CIRCUMSCRIBED.

There are 43 Cases with no deaths.

15 of these gave a typical history and being free of active labyrinthine symptoms were put on the Waiting List for Radical Mastoid operation. All but 4 cases gained complete relief, and in three of these there was some residual vertigo which however was not severe nor persistent enough to require further operation. One patient proceeded to Purulent Labyrinthitis.

5 Cases had active signs and symptoms, four of these requiring bedrest for 2/3 weeks then Radical Operation, while the fifth had an aural polyp removal of which relieved the vertigo and Radical Operation was performed 3 months later.

10 Cases had very poor hearing on account of which early operation was advised: one failed to attend and was lost track of, the other nine having Radical Operation in 4/28 (average 13) days. In 4 the hearing improved as a result, in 4 there is no record.

8 patients required early operation on account of other otitic conditions - facial paralysis in 4, extra-dural abscess in 2, sinus thrombosis, and suspected intracranial tension in others. 1 Case was complicated by Meningitis after Radical Operation.

5 Cases required revision of a previous operation: in one the Radical Operation was revised, in other three there was no useful hearing and Hinsberg Labyrinthotomy was performed to ensure relief of vertigo: in the fourth revision of Radical Operation revealed no fistula and Alcohol Injection was performed.

There were two cases with complication. In one, Diffuse Purulent Labyrinthitis supervened within 48 hours but resolved under Sulphadiazene therapy: there is no record of residual hearing. This case is included under Induced Labyrinthitis also.

The other showed neck rigidity and 2,500 cells per cubic mm. in the spinal fluid the day after operation (which had been performed for relief of facial paralysis), but responded to Sulphadiazine. This case also had no detectable hearing before operation although caloric reactions were present but diminished. This condition was present in 3 other Cases, a finding suggestive of a Circumscribed lesion of the Cochlea, a clinical rarity according to some Authors.

These/

These results would seem to confirm the teaching that routine Radical Operation will deal adequately with most quiescent cases but that operation may be expedited to advantage in cases with poor hearing, and without great danger in other otitic conditions, antibacterial therapy being reserved for possible complications.

There is no indication for Labyrinthectomy unless Radical Operation has failed to relieve the vertigo and there is no useful hearing.

Cases in an active phase require a period of rest until quiescent.

The cases have been grouped as follows:-

Period of Acute Otitis	5 Cases	3 Deaths
Chronic Otitis	7 Cases	6 Deaths
Suppurative	10	
Purulent	15	
Suppurative or Purulent	5	
Complicated	15	

These are five cases secondary to Acute Otitis Media.

- 1 - Suppurative responding to treatment with Carbolic & Glycerine Drops (1934).
- 2 - Purulent with intracranial complication and fatal termination.

In one (1933) function became extinct on the 7th day of the Otitis and Radical Operation was performed but death supervened 2 days later.

MANIFEST LABYRINTHITIS.

It is customary to consider this group under two headings - Serous and Purulent. Some consider "Serous" as a diagnosis in retrospect, i.e. marked interference or even total loss of function which, however, is retained upon recovery: others consider as "Serous" those in which some function, however slight, remains at the time of examination, but as many of these subsequently lose all function and may even suffer complication, they are hardly truly "Serous" in nature.

The former reasoning is probably more correct, but as this correct diagnosis cannot be made at the time all cases are perhaps best considered under the one head of Manifest, although I have separated the "Diagnosis in Retrospect."

The cases have been grouped as follows:-

Result of Acute Otitis 5 Cases 3 Deaths.

" " Chronic Otitis 47 Cases 6 Deaths.

Serous	10	} 47.
Purulent	16	
Serous or Purulent	5	
Complicated	16	

There are five cases secondary to Acute Otitis Media.

- 1 - Serous responding to bedrest with Carbolic & Glycerine Drops (1934).
- 3 - Purulent with introcranial complication and fatal termination.

In one (1933) function became extinct on the 7th day of the Otitis and Hinsberg Operation was performed but death supervened 2 days later.

In/

In the other two Meningitis (C.S.F. Opaque) was present on admission on the 3rd and 7th days respectively, with no hearing or caloric response, but Hinsberg Operation did not avert disaster.

- 1 - Purulent with Meningitis (320 cells) on admission after 5 weeks of bilateral pain and discharge had immediate Neumann Operation (and Cortical on the other ear) and thereafter proseptasine (Haemolytic Streptococci). He made a good recovery.

There are no recent cases of Labyrinthitis as a complication of Acute Otitis, the last being in 1940 in the days of the early Sulphonamides. Unless we are unfortunate enough to have many acute middle ear infections due to virulent organisms which are resistant to all the antibiotics, this group can be relegated to the past.

47 Cases developed in the course of Chronic Otitis.

10 Cases had Manifest symptoms and signs but recovered function, i.e. treatment was successful (Serous).

3 Cases responded to bedrest alone - hearing returned and Radical Operation was performed later, at 6 weeks, 10 weeks and 3 weeks respectively - early in the last case on account of persistent pain.

3 patients failed to respond satisfactorily to bedrest alone, and were submitted to early Radical Operation - at 5, 8 and 14 days, with success. Spinal fluid control was used in one in which function was completely arrested.

2 similar patients had Penicillin and Sulphadiazine respectively after early Radical on 5th day.

2 Cases responded to Penicillin Therapy in addition to bedrest; one had Radical Operation 10 weeks later, but the other after only 48 hours on account of facial paralysis which had become complete, the penicillin being continued post-operatively.

5 others may have recovered function but unfortunately there is no record of whether this was or was not the case.

2 Cases responded to bedrest alone and had later Radical Operation.

2 patients had early Radical Operation, one with C.S.F. control (2 days and 3 days).

1 patient appeared to do well at first but later lost hearing and caloric reaction and had Radical Operation on 25th day.

16 Cases failed to recover function (purulent - uncomplicated).

2 Cases reported in the stage of resolution with diminishing signs and no hearing. No interference and no biotherapy was indicated then and as the chronic otitis cleared up no further action was taken.

8 Cases were submitted to operation without chemo- or bio-therapy. In all cases Hinsberg's Operation was performed. 1 Case reported in the stage of resolution and was allowed to settle for 5 weeks before surgical interference. Increase of spinal cell count in 4 Cases, and loss of hearing or caloric response/

response in the others were used as the actual indication for operation.

4 Cases had both antibacterial therapy and operation, and make a useful picture of the progress of ideas.

1 Case (1938) was submitted to operation as soon as nystagmus to the healthy side appeared. Thereafter Prontosil was administered to prevent complication and L.P. to check on it. Another (1950) was kept under observation without therapy but at rest, yet hearing slowly diminished and finally Radical Operation alone was done, using Penicillin cover to prevent complication and permit Sterilization and healing of the labyrinth, but hearing did not return.

Other two had therapy (one Penicillin and the other Sulphadiazine) during the "Serous" stage but in spite of this hearing diminished and Radical Operation and Labyrinthotomy (respectively) were performed, the former with L.P. control but no return of hearing.

Two patients had Facial Paralysis developing during bedrest and supervision which was otherwise satisfactory, and operation being required Hinsberg's Labyrinthotomy was performed (1934 and 1942) on 10th and 13th day respectively.

16 Cases suffered intracranial complication with total loss of function in the affected ear.

There were 6 Deaths (37.5% mortality).

3 Cases developed Meningitis after early Radical Operation only although function was extinct, and this in spite of post-operative Sulphonamide. 2 responded to Hinsberg's Operation, the third died of Basal Meningitis spreading from the internal meatus.

1 Case with practically total loss of function was controlled for five days with Sulphathiazole and then 5 days with Penicillin before Meningitis developed, but soon after Labyrinthotomy a Cerebellar Abscess became evident. This responded to suboccipital drainage.

12 Cases had Meningitis on admission: five proved fatal. All were subjected to early Labyrinthotomy. 3 Cases were before the Sulphonamide era: 2 with only early invasion recovered, the third, unconscious on admission, died of Cerebellar abscess next day.

after 6 Cases had Prontosil, and 1 Soluseptasine and Sulphathiazole,

after operation, but 4 of the 6 died after steady rise of spinal cell-count, 1 however having a temporal lobe abscess in addition.

2 Cases had Penicillin and Sulphonamides with recovery:-

1 had a temporal-lobe abscess drained per burrhole on the day of admission with systemic and local Sulphanilamide and Penicillin yet proceeded to loss of Labyrinthine function 12 days later when Hinsberg Operation was followed by recovery.

The other had had small doses of both before admission and was continued, after Hinsberg Operation, on full doses of Penicillin and Sulphadiazine, later changed to Streptomycin to which the *B. proteus* was sensitive.

These cases of Manifest Labyrinthitis well exemplify the change in ideas which has taken place since the introduction of Sulphonamides, Penicillin and later Antibiotics.

In a case where some slight function remained (i.e. Serous) it was formerly usual to rely on bed rest and Nature's powers of recuperation, or early Radical Mastoid Operation (followed by frequent spinal punctures to detect invasion) when Nature seemed to be losing the battle. This gave way to post-operative chemo - or bio-therapy to avert the danger of meningitis, but now (with such a wide range of bio-therapy) it would seem better to administer antibiotics from the start and prevent further loss of function, and within about 48 hours relieve the causative factor by Radical Operation under this "umbrella."

As soon as function became extinct (i.e. Serous or Purulent) it was customary to perform an immediate Hinsberg Operation to avert meningeal infection, and the results as regards life were good but at the sacrifice of recoverable hearing in some. Later, repeated lumbar puncture was used to detect an early cell increase in those proceeding to intracranial invasion, this finding or any complication (e.g. facial paralysis) dictating Hinsberg Operation. Again Sulphonamides gave added security after operation.

Still later an attempt was made to limit surgery to the Radical Operation only with the later Sulphonamides and with Penicillin as cover, and has probably resulted in a greater proportion

proportion of healed Labyrinths; and remembering that some cases may still be examples of Serous Labyrinthitis, immediate antibiotic cover and early Radical Operation may even lead to return of function.

The greatest advance, however, has been in the case with intracranial invasion. Formerly early Labyrinthectomy (Neumann Operation) was the only hope, and even with this or Hinsberg Labyrinthotomy and the early Sulphonamides as cover the mortality here was four out of nine cases (See also Induced).

Now, however, we can control Meningitis and even abscess due to the great majority of organisms, and ensure drainage by Hinsberg Operation at the most appropriate moment.

INDUCED LABYRINTHITIS

There are 11 Cases with 2 Deaths (a mortality of 18.2%) but both of these occurred before the advent even of Sulphonamides, in acute exacerbations of chronic otitis media treated by early Radical Operation.

The first occurred in a boy of 8 years (J.Y.) who had also Subperiosteal Abscess formation and aural polypus. Radical Operation the same day showed a perisinus abscess and a large fistula in the lateral canal (There is no note of Labyrinthine symptoms pre-operatively). Two days later there was complete deafness and Nystagmus to the healthy side but the Cerebrospinal fluid was clear and not under tension. Hinsberg Operation showed the stapes to be absent, but three days later lumbar puncture showed turbid fluid containing haemolytic streptococci. Trans-labyrinthine drainage produced no flow of C.S.F. from the internal auditory meatus.

The second case, D.C. of 36 years, was submitted to Radical Operation the day after admission. Within 36 hours there was vomiting, nystagmus to the other (healthy) side and Pyrexia. Re-opening of the cavity showed absence of the stapes and Hinsberg Operation was performed. The spinal fluid then was opalescent and on the next day cloudy, but without tension on each occasion. Death occurred 5 days later.

Of the other cases:-

2 showed only minimal irritation which required no treatment.

3 Cases suffered marked loss of function (Serous Labyrinthitis) and of these, two showed no sign of meningeal involvement and recovery with return of hearing; another also had no signs but was checked by Lumbar Punctures.

4 Cases suffered complete loss of function (Purulent) and 3 were complicated by Meningitis. 2 were controlled by Sulphadiazine alone, one by Hinsberg and Sulphonamide, one by Neumann and Sulphanilamide.

There is thus no indication for Hinsberg Operation before loss of function, since any Meningitis can be controlled by chemo- or bio-therapy provided the infecting organism is sensitive. Nor does a spinal cell increase before loss of function determine operation, nor total loss of function with no cell increase (Lund 1936).

It would seem better to give maximum therapy as early as possible and watch the spinal fluid should total loss of function occur, any serious rise determining Radical Operation only on the chance of return of function after Serous Labyrinthitis or at least of a healed and safe labyrinth.

Facial paralysis occurred in 3 cases. In one it was of only 5 days' duration and recovered after Radical Operation only (although a large fistula was found in the lateral canal), while another of a year's standing recovered after removal of a vegetation of the cochlea and decompression. Meningitis complicated a further two cases. One an acute exacerbation improved slightly after Radical Operation but required Labyrinth drainage and fresh blood transfusion for final recovery. The other, also with facial paralysis, recovered on systemic and intrathecal penicillin. Revision of Radical Operation one week later revealed a large encapsulation of Petrous Apex with O.E.S. leak, the labyrinth supposedly being repaired.

These may be placed therefore as:-

Repaired 3
Occasional 1 at least.
Fistulae 10
Complicated 4

Treatment

LATENT LABYRINTHITIS

There are 18 Cases with no deaths.

In five of these there were recurrent polypi which, in the presence of a non-functioning labyrinth, required Radical Mastoid operation at which a large fistula or granulations of the inner ear dictated Hinsberg Labyrinthotomy.

In one case with recurrent polypi Radical Operation alone was performed although fistulae were found in the roof of the vestibule and the posterior end of the vertical canal, and progress was uneventful.

In four Cases there was an acute exacerbation of chronic mastoiditis and a dead labyrinth, and after 2/7 days' bed-rest with improvement Radical Operation showed an unhealed labyrinth which required drainage (Hinsberg or enlargement of fistula).

Four others underwent routine Radical Operation for chronic Mastoiditis but destruction and fistulae were thought to require exenteration of a non-functioning inner ear, although in one at least of these there was evidence of healing by ossification.

Facial paralysis complicated two other Cases. In one it was of only 5 days' duration and recovered after Radical Operation only (although a large fistula was found in the lateral canal), while another of 1 year's standing recovered after removal of a sequestrum of the cochlea and decompression. Meningitis complicated a further two Cases. One an acute exacerbation improved slightly after Radical Operation but required Labyrinth drainage and fresh blood transfusion for final recovery. The other, also with facial paralysis, recovered on systemic and intrathecal penicillin: revision of Radical Operation one week later revealed a large cholesteatoma of Petrous Apex with C.S.F. leak, the labyrinth apparently being repaired.

These may be classed therefore as:-

Repaired	3
Ossified	1 at least.
Fistulous	10
Complicated	4

Treatment/

Treatment would seem to depend on the findings at Radical Operation; persistent fistula and granulation of the inner ear requiring exenteration, while an intact capsule or healthy fistula is better left alone.

No chemo- or biotherapy seems indicated unless there is post-operative complication, and this occurred in only one case - recurrent polypi treated by Hinsberg Operation and Amnioplastin graft, the cavity however being never really satisfactory. Eight months later Meningitis (300 cells, no organisms) supervened but was controlled with Sulphapyridine.

COMPARISON OF RESULTS.

In order fully to appreciate the impact of the Sulphonamides and of Penicillin upon Labyrinthitis and its consequences, these cases must be grouped into the period during which these drugs were available.

	Before Sulphonamides	Sulphonamides available	Penicillin available
Number of cases.	49	50	25
" " " of manifest Labyrinthitis	21	30	12
Percentage of Deaths	14.3	8	-
" " Complications	52.4	40	33.3
" " complications after admission	19	3.3	4.0
" " Manifest recovered function	25	30	42

The elimination of mortality and the increase in the proportion who recovered function are obvious. That the complications after admission with Penicillin should be higher than with Sulphonamides is due to one case who received only 25,000 units 3 hourly (cf Huber, page 24) but proved later to have a cerebellar abscess.

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